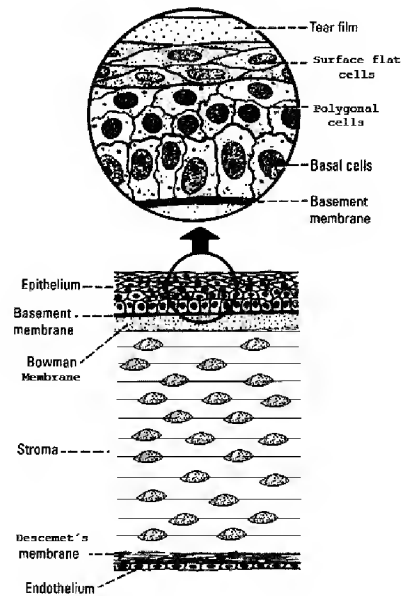
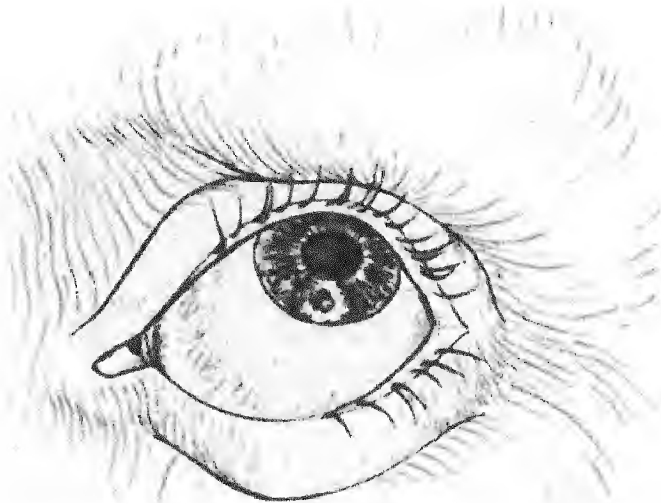


# Cornea



***Dr/ Mahmoud Medhat***

*To the only one who gave me **HOPE***

The cornea is the avascular, transparent, clear & lustrous anterior 1/6 of the outer protective coat of the eye.

It is the main refractive surface of the eye (42 D).

### **Minute anatomy:**

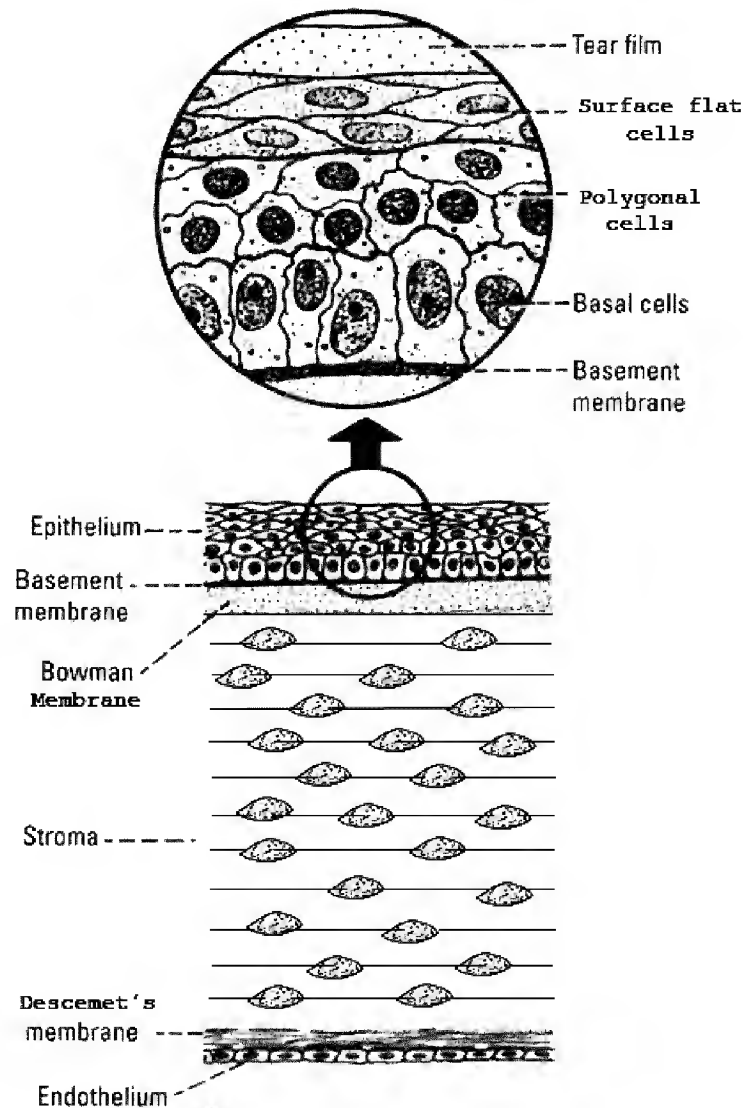
1. **Epithelium:** Stratified squamous non-keratinized epithelium. 5-6 layers thick.
2. **Bowman's membrane:** Structureless acellular elastic membrane. If destroyed, it never regenerates.
3. **Stroma:** Forms the main thickness of the cornea. It consists of 100-150 regular collagen fibrils.
4. **Descemet's membrane:** Elastic membrane which easily regenerates.
5. **Endothelium:** Single layer of hexagonal cells, responsible for corneal dehydration. This layer never regenerates.

**N.B.** Corneal ulcers are ocular emergencies.

**N.B.** Any corneal ulcer is considered infective until proved otherwise.

### **Factors protecting the corneal surface:**

1. **Tears:**
  - a. Protect the cornea from dryness.
  - b. Contain lysozymes, which has antimicrobial action.
  - c. Supply the cornea with O<sub>2</sub> & nutrients.
2. **Lids:**
  - a. Closure of the lids protects against exposure.
  - b. Blinking is essential for tear film distribution.
3. **High sensitivity:** Irritation of the corneal nerves causes reflex lacrimation & blinking.
4. **Rapid turnover of the corneal epithelium.**



## Infective corneal ulcer

### I. Bacterial corneal ulcer:

#### Predisposing factors

- |                            |                       |
|----------------------------|-----------------------|
| 1. Corneal abrasion.       | 4. Contact lens wear. |
| 2. Corneal anaesthesia.    | 5. Dry eye.           |
| 3. Blepharoconjunctivitis. | 6. Exposure.          |

**Causative organism:** High virulent organisms can invade the cornea without the aid of any predisposing factor, like:

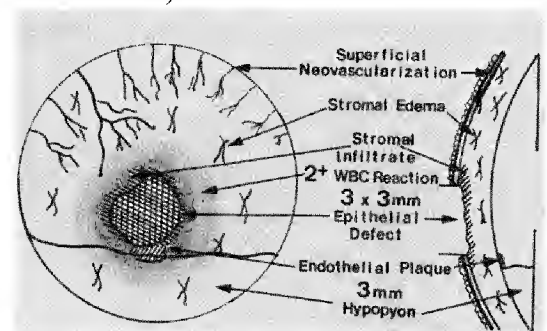
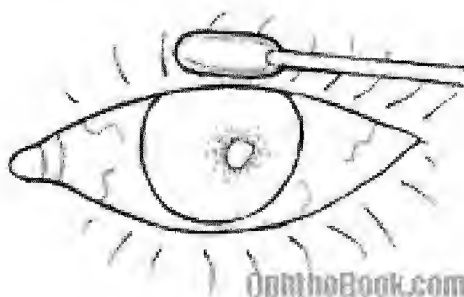
- |                    |                            |
|--------------------|----------------------------|
| 1. N. gonorrhea.   | 3. Listeria.               |
| 2. C. diphtheriae. | 4. Haemophilus aegypticus. |

#### Pathology of corneal ulcers

1. **Progressive stage:** Entrance of the organism through the epithelium ⇒ toxins & enzymes tissue ⇒ infiltration & necrosis & sloughing ⇒ ulcer.
2. **Regressive stage:** ↓↓ infiltration, sloughing of the necrotic material. The floor becomes clear with a well-demarcated edge. The cornea is invaded by blood vessels.
3. **Healing stage:** Epithelial sliding and multiplication ⇒ covering of the ulcer. The blood vessels become thin & empty (Ghost blood vessels).

#### Symptoms

1. Pain, photophobia, blepharospasm & reflex lacrimation.
2. Decresed vision (If the central cornea is involved).



## **Signs**

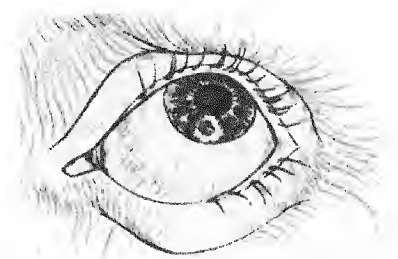
1. Edema of the lids & conjunctiva.
2. Loss of the corneal luster.
3. Infiltration in the edge & bed of the corneal ulcer.
4. Staining with fluorescein solution shows the ulcer.
5. Anterior chamber reaction: Flare, cells & even hypopyon.
6. Acute serpiginous ulcer (Hypopyon ulcer):
  - a. Central ulcer, typically caused by Pneumococci.
  - b. Undermined central advancing edge & sloping peripheral healing edge.
  - c. Hypopyon formation.

## **Complications**

1. Anterior uveitis: Hypopyon & posterior synechiae may form.
2. Progression: In depth &/or area.
3. Perforation.
4. Corneal opacity.

## **Complications of perforated corneal ulcer**

1. Intraocular haemorrhage, Subluxation & dislocation of the lens:  
Due to sudden lowering of the IOP.
2. Endophthalmitis.
3. Leucoma adherent.
4. Peripheral anterior synechiae: Perforation ⇒ flattening of the anterior chamber ⇒ the cornea becomes adherent to the anterior surface of the iris. Early the adhesions are fibrinous, while late become fibrous & permanent.
5. Corneal fistula: Migration of the corneal epithelium to line the ulcer.
6. Epithelialization: Furthermore migration of the corneal epithelium to line the back of the cornea, the anterior chamber angle & the anterior surface of the iris.
7. Secondary glaucoma.
8. Complicated cataract.
9. Anterior staphyloma.



## **Differential diagnosis (Fungal & acanthamoeba Keratitis)**

|                             | <b>Fungal ulcers</b>                                                                                                                                                                                                                      | <b>Acanthameba ulcers</b>                                                                                                                                                                                                            |
|-----------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Predisposing factors</b> | Trauma with objects of plant sources.                                                                                                                                                                                                     | Contact lens wear, with improper cleaning.                                                                                                                                                                                           |
| <b>Characters</b>           | <ul style="list-style-type: none"> <li>▪ Dry, gray lesion. Somewhat elevated.</li> <li>▪ Stromal infiltration with feathery edges.</li> <li>▪ Satellite lesions, immune ring, endothelial plaques &amp; thick cheesy hypopyon.</li> </ul> | <ul style="list-style-type: none"> <li>▪ Severe pain.</li> <li>▪ Punctate erosions &amp; broad dendrites.</li> <li>▪ Crescent or annular infiltration with satellites, immune ring or double ring &amp; thickened nerves.</li> </ul> |
| <b>Diagnosis</b>            | <ul style="list-style-type: none"> <li>▪ Cultures on special media.</li> <li>▪ Corneal biopsy.</li> <li>▪ Confocal scanning microscope examination.</li> </ul>                                                                            |                                                                                                                                                                                                                                      |
| <b>Treatment</b>            | <ul style="list-style-type: none"> <li>▪ antifungal drugs: Amphotericin-B</li> <li>▪ Systemic antifungal drugs.</li> <li>▪ Therapeutic keratoplasty.</li> </ul>                                                                           | <ul style="list-style-type: none"> <li>▪ Diamidines, neomycin, ketoconazole, &amp; clotrimazole.</li> <li>▪ Therapeutic keratoplasty.</li> </ul>                                                                                     |

## **Treatment of corneal ulcers**

### **1. Antibiotics:**

- Local broad spectrum antibiotics:
  - Ciprofloxacin: Covering nearly all organisms.
  - Fortified aminoglycosides & cephalosporines.
- Subconjunctival & systemic antibiotics.

### **2. Patching:** Protects the newly formed epithelium.

### **3. Bandage contact lens.**

### **4. Cycloplegic:** ↓↓ pain (relieves cyclospasm) & prevents posterior synechiae.

### **5. Surgery:**

- a. Adhesive glue: To seal small perforations.
- b. Paracentesis: Indicated in cases of descemetocoele (Impending perforation) & Hypopyon with secondary glaucoma.
- c. Conjunctival flap & lateral tarsorrhaphy: Promote healing.
- d. Corneal grafting: for treatment of corneal opacities.

## **II. Viral corneal ulcer:**

### **A. Herpes simplex Keratitis:**

**Causative organism:** Herpes simplex virus type I.

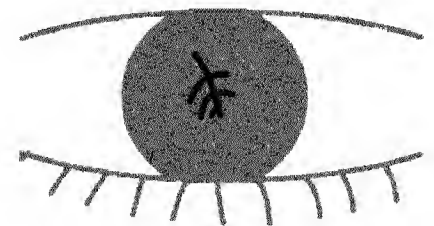
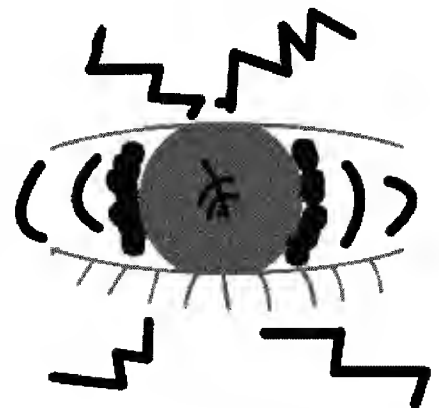
#### **Pathology**

- Primary H. simplex Keratitis: Widespread superficial lesions.
- After the primary infection the virus remains latent in the sensory ganglia.
- Reactivation of the dormant focus: Steroids, stress or debilitating diseases.
- Recurrent H. simplex Keratitis: More localized corneal lesion & the stroma may show inflammatory cellular reaction (Due to presence of Ab's against HSV).

#### **Signs**

Dendritic ulcer:

- Central, linear & branching infiltration ending in rounded knobs.
- Shedding of the infected epithelium ⇒ dendritic ulcer.
- The ulcer bed stains with fluorescein, while the diseased epithelium stains with rose Bengal.
- The ulcer is superficial & non-vascularized.  
The bed of the ulcer is not sensitive.



Other forms of herpetic Keratitis:

- Geographic (Amoeboid): With low immunity or use of steroids.
- Deepening of the ulcer.
- Disciform Keratitis: Ag/Ab reaction.
- Deep necrotizing Keratitis: Actual stromal invasion by the virus.
- Limbal ulcer.

## Treatment

1. **Antiviral drugs:** Acyclovir, Vidarabine, T3F, Idoxouridine.
2. Patching.
3. Bandage contact lens.
4. Cycloplegic: ↓↓ pain (relieve cyclospasm) & prevent posterior synechiae.
5. Surgery:
  - a. Debridement of the infected epithelium.
  - b. Penetrating keratoplasty for opacified corneas.

N.B. Steroids are absolutely contraindicated.

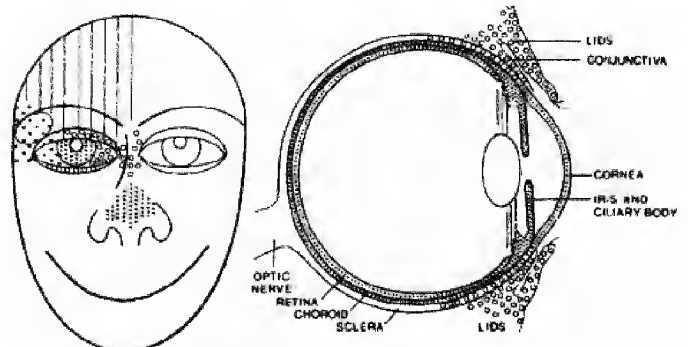
## **B. herpes Zoster Ophthalmicus (HZO):**

### Clinical picture

1. **Prodroma:** Fever, headache, malaise & neuralgic pain along distribution of the nerve affected.
2. **Skin lesions:** Papules ⇒ pustules ⇒ crusting ulcers ⇒ healing by punched out scars.

### 3. **Ocular lesions:**

- MPC, Episcleritis & Scleritis.
- Anterior uveitis & acute retinal necrosis.
- Corneal lesions:  
Punctate Epithelial  
Keratitis, nummular  
Keratitis, disciform Keratitis & microdendrites.



| KEY |                 |  |                                               |
|-----|-----------------|--|-----------------------------------------------|
|     | FRONTAL BRANCH  |  | INFRATROCHLEAR DIVISION OF NASOCILIARY BRANCH |
|     | LACRIMAL BRANCH |  | NASAL DIVISION OF NASOCILIARY BRANCH          |

4. **Neurological:** Encephalitis & cranial nerve affection.

## Treatment

1. Systemic acyclovir.
2. Skin lesions: Antiviral & steroid-antibiotic cream.
3. Ocular lesions:
  - a. Antiviral drugs.
  - b. Patching.
  - c. Bandage contact lens.
  - d. Corneal grafting for corneal opacities.
  - e. Corticosteroids.

## **Non-Infective corneal ulcer**

Neuroparalytic ulcer.

Neuropathic ulcer.

Keratomalacia.

Photophthalmia.

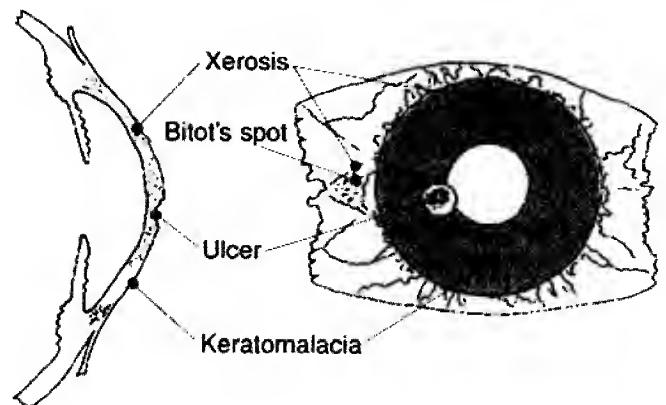
## **Keratomalacia**

Acute melting of the cornea, due to vitamin A deficiency.

**Etiology:** Advanced starvation & marasmus in very young children.

## **Clinical picture**

1. Bilateral melting of the cornea.
2. Minimal inflammatory response.
3. Prolapse of the ocular contents.
4. 2ry bacterial infection & panophthalmitis.
5. Blindness.



## **Management**

1. Large doses of systemic & local vitamin A.
2. Improve general condition.



## Keratoconus

**Definition:** Progressive central stromal thinning  $\Rightarrow$  ectasia & apical protrusion.

### Pathology

- Starts around puberty (10-20 years) and progresses for few years.
- Bilateral in 85% of cases.
- Bilateral cases are usually asymmetrical.
- Sometimes associated with:
  - Systemic diseases: Down's syndrome, Marfan's syndrome.
  - Ocular diseases: Spring catarrh & retinitis pigmentosa.



**Symptoms:** Frequent change of glasses, due to rapidly progressive myopia & astigmatism.

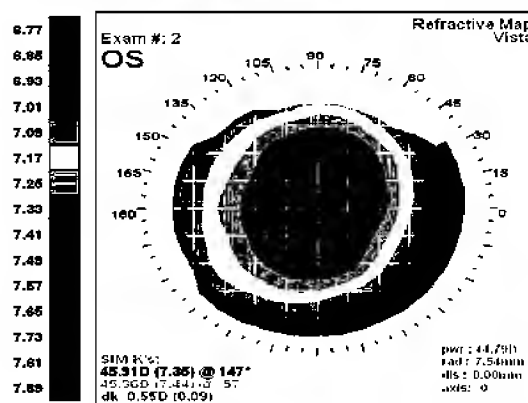
### Early signs

1. **Retinoscopy:** Spinning reflex.
2. **Keratometry:** Irregular mires.
3. **Placido disc:** Irregular circles with central crowding.
4. **Corneal topography:** For diagnosis of subclinical cases.

### Late

1. **Localized area of corneal thinning.**
2. **Apical protrusion**  $\Rightarrow$  cone-shaped deformity.
3. **Deep anterior chamber.**
4. **Munson sign:** Downward gaze  $\Rightarrow$  protrusion of the lower lid.
5. **Fleisher ring:** Iron deposits at the base of the cone.
6. **Vogt striae:** Indicating early opacity.

**N.B. Acute hydrops:** Continuous stretch & ectasia of the cornea leads to breaks in DM, causing acute corneal edema & opacification. The condition resolves in few weeks, leaving residual central opacity.



## **Management**

1. Early cases before irregular astigmatism: Spectacles.
2. With irregular astigmatism: Hard contact lens.
3. Surgery: Penetrating keratoplasty as the final line of treatment.

Less common surgeries:

- Epikeratophakia.
- Deep lamellar keratoplasty.
- Intrastromal corneal rings.

**N.B.** Keratorefractive procedures are contraindicated in keratoconus as they make the case worse, so early diagnosis of keratoconus is essential.

## **Corneal opacities**

### **Mechanism of affecting vision:**

#### **1. Dense central leukoma:**

- Blocks the passage of light rays.

- Dense peripheral scar may not affect vision, unless it affects the corneal curvature  $\Rightarrow$  astigmatism.
  - Leukoma adherent may be associated with 2ry glaucoma.
2. **Nebulae:** This faint opacity does not block the passage of light, but it scatters the rays in irregular pattern causing affection of vision, even by paracentral nebula & clear central cornea.

## Management

1. Central leukoma: Penetrating keratoplasty.
2. Peripheral scars:
  - Glasses.
  - Contact lenses: For astigmatism &/or cosmetically.
  - Surgical treatment.
3. Nebulae causing irregular astigmatism:
  - Contact lens.
  - Excimer laser.
  - Lamellar keratoplasty.

## Keratoplasty

Replacement of the diseased corneal tissue by healthy tissue.

### Source:

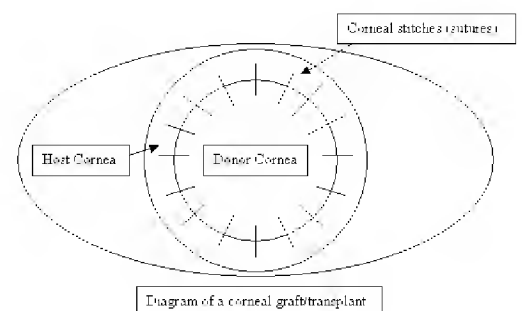
- Autogenous graft: From the same patient.
- Allograft: From cadaver eyes.

### Types:

- Penetrating: Full thickness corneal graft.
- Lamellar: Partial thickness graft.

### Indications:

- Corneal opacities.
- Keratoconus.
- Corneal perforation or fistula.
- Resistant corneal ulcer, especially fungal & acanthamoeba.



## **Episcleritis**

Inflammation of the episcleral tissue, which is more common in females.

### **Etiology**

1. Idiopathic: Most common.
2. Allergy to endogenous toxin:
  - Septic focus.
  - Tuberculous focus.
3. Collagen diseases: e.g. rheumatoid arthritis.

### **Symptoms**

1. Discomfort.
2. Lacrimation.
3. Tenderness on pressing the globe.

### **Signs**

| <b>Nodular type</b>                                                                                                                                                                                                       | <b>Diffuse type</b>                                                                                                                |
|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------|
| <ol style="list-style-type: none"> <li>1. Lentil size, well circumscribed.</li> <li>2. purple in colour, due to dilatation of the deep vessels.</li> <li>3. Tender &amp; the conjunctiva moves freely over it.</li> </ol> | <ol style="list-style-type: none"> <li>1. Dilated episcleral vessels.</li> <li>2. Frequently affecting only one sector.</li> </ol> |

**Differential diagnosis:** Should be differentiated from phlycten.

### **Treatment**

1. Topical steroids e.g. prednisolone & dexamethazone.
2. Oral NSAIDs e.g. phenylbutazone.

## **Scleritis**

Inflammation affecting all layers of the sclera, which is more serious than episcleritis.

### **Etiology**

1. Associated with rheumatoid arthritis: In 50% of cases.
2. Idiopathic in 50% of cases.

### **Symptoms**

1. Severe pain.
2. Photophobia.
3. Lacrimation.

### **Signs**

| <b>Anterior type</b>                                                                                                                                                                                                                                                    | <b>Posterior type</b>                                          |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------|
| <ol style="list-style-type: none"><li>1. Nodular: Deep nodule, fixed to the sclera.</li><li>2. Diffuse.</li><li>3. Annular.</li><li>4. Necrotizing: May cause rupture of the globe.</li><li>5. Scleromalacia perforans: Melting of the sclera in a quiet eye.</li></ol> | Causes severe pain, uveitis with exudative retinal detachment. |

### **Complications**

1. Uveitis.
2. Sclerosing keratitis: Opacity developing in the cornea near the scleral nodule.
3. Scleral staphyloma.

### **Treatment**

1. Topical steroids: Usually ineffective.
2. Oral NSAIDs.
3. Large doses of oral steroids (1-2 mg/kg/day), immunosuppressives.